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Exhibit I

L3 ANSWER 12 OF 21 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 1999190498 MEDLINE
DOCUMENT NUMBER: 99190498 PubMed ID: 10092077
TITLE: Cloning of murine NKG2A, B and C: second family of C-type lectin receptors on murine NK cells.
AUTHOR: Lohwasser S; Hande P; Mager D L; Takei F
CORPORATE SOURCE: Terry Fox Laboratory, British Columbia Cancer Agency, Vancouver, Canada.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1999 Mar) 29 (3) 755-61.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF109782; GENBANK-AF109783; GENBANK-AF109784; GENBANK-AF109785
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990504
Last Updated on STN: 19990504
Entered Medline: 19990421

AB Multiple NK cell receptors for MHC class I have been identified. They include killer inhibitory receptors and CD94/NKG2 heterodimers in humans and the Ly49 family in mice. Here we report the cloning of murine NKG2A, B and C. The deduced amino acid sequence of mouse NKG2A contains only one consensus cytoplasmic immunoreceptor tyrosine-based inhibitory motif (ITIM). NKG2A from B6 and BALB/c mice differ by six amino acid residues in the extracellular domain. Murine NKG2B, like its human conterpart, appears to be a **splice variant** of NKG2A and lacks a large portion of the stalk region. Murine NKG2C lacks an ITIM in its cytoplasmic domain, a feature shared by human and rat NKG2C. However, unlike the human counterpart, the transmembrane domain of mouse NKG2C does not contain a charged amino acid residue. Mouse NKG2A mRNA was detected in IL-2-activated NK cells and spleen cells but not in other tissues. The NKG2A gene was localized on the distal portion of chromosome 6 where the NK complex has been located. These results further extend the repertoire of C-type **lectin** receptors on murine NK cells.

L3 ANSWER 9 OF 21 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2001469022 MEDLINE
DOCUMENT NUMBER: 21405030 PubMed ID: 11513955
TITLE: Molecular characterization of two novel alternative spliced
variants of the KLRF1 gene and subcellular distribution of KLRF1 isoforms.
AUTHOR: Roda-Navarro P; Hernanz-Falcon P; Arce I; Fernandez-Ruiz E
CORPORATE SOURCE: Unidad de Biologia Molecular, Hospital Universitario de la Princesa, Universidad Autonoma de Madrid, C/Diego de Leon 62, 28006, Madrid, Spain.
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (2001 Aug 30) 1520 (2) 141-6.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF267244; GENBANK-AF267245
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010830
Last Updated on STN: 20010917
Entered Medline: 20010913

AB The killer cell **lectin**-like receptor (KLR) family is formed by type II transmembrane glycoproteins with a single extracellular C-type **lectin**-like domain (CTLD). Some of these glycoproteins are involved in the regulation of natural killer cell activity. Recently, we have described the molecular characterization of the KLRF1 gene and the existence of one alternative spliced form, lacking the stalk region of the extracellular domain. In this work we describe two novel KLRF1 alternative **spliced variants** coding for truncated proteins lacking the CTLD. In addition, we present the biochemical analysis of the KLRF1 protein and the subcellular distribution of all KLRF1 isoforms expressed in heterologous transfectants.

L3 ANSWER 8 OF 21 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 2001143205 MEDLINE
DOCUMENT NUMBER: 21115136 PubMed ID: 11220622
TITLE: Genomic structure, alternative splicing, and physical mapping of the killer cell lectin-like receptor G1 gene (KLRG1), the mouse homologue of MAFA.
AUTHOR: Voehringer D; Kaufmann M; Pircher H
CORPORATE SOURCE: Institute for Medical Microbiology and Hygiene, Department of Immunology, University of Freiburg, Germany.
SOURCE: IMMUNOGENETICS, (2001) 52 (3-4) 206-11.
Journal code: 0420404. ISSN: 0093-7711.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010308

AB The mouse killer cell **lectin**-like receptor G1 (KLRG1), the mouse homologue of the mast cell function-associated antigen (MAFA), is an inhibitory C-type **lectin** expressed on natural killer (NK) cells and activated CD8 T cells. Here we report the complete nucleotide sequence, alternatively **spliced variants**, and the physical mapping of the KLRG1 gene in the mouse. The gene spans about 13 kb and consists of five exons. Short interspersed repeats of the B1 and B2 family, a LINE-1-like element, and a (CTT)170 triplet repeat were found in intron sequences. In contrast to human KLRG1 and to the murine KLR family members, mouse KLRG1 locates outside the NK complex on Chromosome 6 between the genes encoding CD9 and CD4.

L5 ANSWER 171 OF 171 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1990:482551 BIOSIS
DOCUMENT NUMBER: BR39:106572
TITLE: THE BREAST **CANCER** ASSOCIATED MUCIN MAM-6 IS GENERATED BY A POLYMORPHIC GENE ENCODING **SPLICE VARIANTS** WITH TWO ALTERNATIVE AMINO TERMINI.
AUTHOR(S): LIGTENBERG M J L; GENNISSEN A M C; VOS H L; HILKENS J
CORPORATE SOURCE: DEP. TUMOR BIOL., NETHERLANDS CANCER INST., PLESMANLAAN 121, 1066 CX AMSTERDAM, NETHERLANDS.
SOURCE: SYMPOSIUM ON MOLECULAR, BIOCHEMICAL, AND CELLULAR BIOLOGY OF HUMAN BREAST CANCER HELD AT THE 19TH ANNUAL UCLA (UNIVERSITY OF CALIFORNIA-LOS ANGELES) SYMPOSIA ON MOLECULAR AND CELLULAR BIOLOGY, TAMARRON, COLORADO, USA, FEBRUARY 3-8, 1990. J CELL BIOCHEM SUPPL, (1990) 0 (14 PART B), 338.
CODEN: JCBSD7.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L5 ANSWER 168 OF 171 CANCERLIT
ACCESSION NUMBER: 94690762 CANCERLIT
DOCUMENT NUMBER: 94690762
TITLE: C-src expression in neuroendocrine **tumors**: neuronal **splice variants** of c-src as diagnostic and prognostic markers in neuroblastoma.
AUTHOR: Bjelfman C
CORPORATE SOURCE: Uppsala Universitet, Sweden.
SOURCE: Diss Abstr Int [C], (1993). Vol. 54, No. 1, pp. 222. ISSN: 0419-4217.
DOCUMENT TYPE: (THESIS)
FILE SEGMENT: ICDB
LANGUAGE: English
ENTRY MONTH: 199409

L5 ANSWER 164 OF 171 MEDLINE DUPLICATE 86

ACCESSION NUMBER: 94090349 MEDLINE
DOCUMENT NUMBER: 94090349 PubMed ID: 8266105
TITLE: WT1-mediated growth suppression of Wilms **tumor** cells expressing a WT1 **splicing variant**
.
AUTHOR: Haber D A; Park S; Maheswaran S; Englert C; Re G G; Hazen-Martin D J; Sens D A; Garvin A J
CORPORATE SOURCE: Laboratory of Molecular Genetics, Massachusetts General Hospital Cancer Center, Boston 02129.
CONTRACT NUMBER: CA37887 (NCI)
CA58596 (NCI)
SOURCE: SCIENCE, (1993 Dec 24) 262 (5142) 2057-9.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401
ENTRY DATE: Entered STN: 19940209
Last Updated on STN: 20000303
Entered Medline: 19940121

L5 ANSWER 154 OF 171 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1994:291416 BIOSIS
DOCUMENT NUMBER: PREV199497304416
TITLE: Mutations in the NF2 gene transcript in multiple **tumor** types. Identification of an alternative **splice variant** and characterization of the merlin gene product.
AUTHOR(S): Bianchi, A. B.; Hara, T.; Ramesh, V.; Klein-Szanto, A.; Gusella, J.; Lekanne-Deprez, R.; Zwarthoff, E.; Seizinger, B. R.; Kley, N.
CORPORATE SOURCE: Dep. Mol. Genetics Cell Biol., Bristol-Myers Squibb Pharm. Res. Inst., Princeton, NJ 08543 USA
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (1994) Vol. 35, No. 0, pp. 610.
Meeting Info.: 85th Annual Meeting of the American Association for Cancer Research San Francisco, California, USA April 10-13, 1994
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 150 OF 171 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1995:241556 BIOSIS

DOCUMENT NUMBER: PREV199598255856

TITLE: The identification of a novel amino-terminal **splice variant** of Pit-1 in human non-functioning pituitary **tumours**.

AUTHOR(S): Ball, S. G. (1); Carroll, R. S.; Zhang, Jianping; Black, P. M.; Chin, W. W.

CORPORATE SOURCE: (1) HHMI, Div. Genet., Dep. Med., Brigham and Women's Hosp., Boston, MA 02115 USA

SOURCE: Journal of Endocrinology, (1995) Vol. 144, No. SUPPL., pp. RC9.
Meeting Info.: 14th Joint Meeting of the British Endocrine Societies and the European Federation of Endocrine Societies Warwick, England, UK March 27-30, 1995
ISSN: 0022-0795.

DOCUMENT TYPE: Conference

LANGUAGE: English

L5 ANSWER 143 OF 171 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:185193 BIOSIS
DOCUMENT NUMBER: PREV199598199493
TITLE: Two different 3'**splice variants** of PDGF B in human **tumor** cells.
AUTHOR(S): Heller, S.; Scheibenpflug, L.; Westermark, B.; Nister, M.
CORPORATE SOURCE: Dep. Pathology, Univ. Hosp., S-751 85 Uppsala Sweden
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (1995) Vol. 36, No. 0, pp. 169.
Meeting Info.: Eighty-sixth Annual Meeting of the American Association for Cancer Research Toronto, Ontario, Canada March 18-22, 1995
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 141 OF 171 MEDLINE DUPLICATE 77
ACCESSION NUMBER: 96341739 MEDLINE
DOCUMENT NUMBER: 96341739 PubMed ID: 8750183
TITLE: Schwann cell **tumors** express characteristic patterns of CD44 **splice variants**.
AUTHOR: Sherman L; Skroch-Angel P; Moll J; Schwechheimer K; Ponta H; Herrlich P; Hofmann M
CORPORATE SOURCE: Institut fur Genetik, Kernforschungszentrum Karlsruhe, Germany.
SOURCE: JOURNAL OF NEURO-ONCOLOGY, (1995 Dec) 26 (3) 171-84. Ref: 57
Journal code: 8309335. ISSN: 0167-594X.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961022
Last Updated on STN: 19970203
Entered Medline: 19961008

L5 ANSWER 126 OF 171 MEDLINE DUPLICATE 64
ACCESSION NUMBER: 96027605 MEDLINE
DOCUMENT NUMBER: 96027605 PubMed ID: 7559633
TITLE: A kinase-deficient **splice variant** of the human JAK3 is expressed in hematopoietic and epithelial **cancer** cells.
AUTHOR: Lai K S; Jin Y; Graham D K; Witthuhn B A; Ihle J N; Liu E T
CORPORATE SOURCE: Department of Biology, Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill 27599-7295, USA.
CONTRACT NUMBER: P50-CA58223-03 (NCI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Oct 20) 270 (42) 25028-36.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U31317; GENBANK-U31601; GENBANK-U31602
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951121

L7 ANSWER 14 OF 28 MEDLINE
ACCESSION NUMBER: 1999455126 MEDLINE
DOCUMENT NUMBER: 99455126 PubMed ID: 10523680
TITLE: Xenografts of human solid tumors frequently express cellular-associated isoform of vascular endothelial growth factor (VEGF) 189.
AUTHOR: Okamoto K; Oshika Y; Fukushima Y; Ohnishi Y; Tokunaga T; Tomii Y; Kijima H; Yamazaki H; Ueyama Y; Tamaoki N; Nakumura M
CORPORATE SOURCE: Department of Pathology, Tokai University School of Medicine, Bohseidai, Isehara-shi, Kanagawa 259-1193, Japan.
SOURCE: ONCOLOGY REPORTS, (1999 Nov-Dec) 6 (6) 1201-4. Journal code: 9422756. ISSN: 1021-335X.
PUB. COUNTRY: Greece
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991207

AB Vascular endothelial growth factor (VEGF), a major factor mediating tumor stromal angiogenesis, is expressed as five **splice variants** encoded by a single gene (VEGF121, VEGF145, VEGF165, VEGF189 and VEGF206). Recently, we demonstrated that the cell-associated isoform, VEGF189, plays important roles in establishment of human colon and esophageal cancer xenografts. We have established 228 xenografts originating from various human solid tumors. In this study, we investigated the expression patterns of VEGF isoforms in those tumor xenografts by RT-PCR. The isoform patterns were VEGF121/VEGF165 in 27 xenografts (11.8%) and VEGF121/VEGF165/VEGF189 in 201 (88.2%). All human solid tumor xenografts expressed VEGF189 more frequently than primary tumors reported previously. These results suggest that VEGF189 contributes to the successful xenotransplantability of various human solid tumors via augmentation of stromal vascularization.

L7 ANSWER 11 OF 28 MEDLINE
ACCESSION NUMBER: 2000206935 MEDLINE
DOCUMENT NUMBER: 20206935 PubMed ID: 10739878
TITLE: Modification of alternative splicing pathways as a potential approach to chemotherapy.
AUTHOR: Mercatante D; Kole R
CORPORATE SOURCE: Lineberger Comprehensive Cancer Center and Department of Pharmacology, University of North Carolina, CB 7295, Chapel Hill, NC, USA.
SOURCE: PHARMACOLOGY AND THERAPEUTICS, (2000 Mar) 85 (3) 237-43. Ref: 51
Journal code: 7905840. ISSN: 0163-7258.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000706
Last Updated on STN: 20000706
Entered Medline: 20000628

AB Many cancer-associated genes are alternatively spliced; their expression leads to the production of multiple **splice variants**. Although the functions of most of these variants are not well-defined, some have antagonistic activities related to regulated cell death mechanisms. In a number of cancers and cancer cell lines, the ratio of the **splice variants** is frequently shifted so that the anti-apoptotic **splice variant** predominates. This observation suggests that modification of splicing, which restores the proper ratio of alternatively spliced gene products, may reverse the malignant phenotype of the cells and offer a gene-specific form of anticancer chemotherapy. Our laboratory has extensively investigated the use of antisense oligonucleotides for shifting the splicing patterns of several genes. Potential application of this method for treatment of cancers, as well as of certain genetic disorders, is discussed.

L7 ANSWER 12 OF 28 MEDLINE

L7 ANSWER 9 OF 28 MEDLINE
ACCESSION NUMBER: 2000484196 MEDLINE
DOCUMENT NUMBER: 20442415 PubMed ID: 10962031
TITLE: Isolation and sequencing of cDNAs for **splice variants** of growth hormone-releasing hormone receptors from human cancers.
AUTHOR: Rekasi Z; Czompoly T; Schally A V; Halmos G
CORPORATE SOURCE: Endocrine, Polypeptide and Cancer Institute, Veterans Affairs Medical Center, Department of Medicine, Tulane University School of Medicine, New Orleans, LA 70112, USA.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Sep 12) 97 (19) 10561-6. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF282259; GENBANK-AF282260; GENBANK-AF282261; GENBANK-AF282262
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001012

AB The proliferation of various tumors is inhibited by the antagonists of growth hormone-releasing hormone (GHRH) in vitro and in vivo, but the receptors mediating the effects of GHRH antagonists have not been identified so far. Using an approach based on PCR, we detected two major **splice variants** (SVs) of mRNA for human GHRH receptor (GHRH-R) in human cancer cell lines, including LNCaP prostatic, MiaPaCa-2 pancreatic, MDA-MB-468 breast, OV-1063 ovarian, and H-69 small-cell lung carcinomas. In addition, high-affinity, low-capacity binding sites for GHRH antagonists were found on the membranes of cancer cell lines such as MiaPaCa-2 that are negative for the vasoactive intestinal peptide/pituitary adenylate cyclase-activating polypeptide receptor (VPAC-R) or lines such as LNCaP that are positive for VPAC-R. Sequence analysis of cDNAs revealed that the first three exons in SV(1) and SV(2) are replaced by a fragment of retained intron 3 having a new putative in-frame start codon. The rest of the coding region of SV(1) is identical to that of human pituitary GHRH-R, whereas in SV(2) exon 7 is spliced out, resulting in a 1-nt upstream frameshift, which leads to a premature stop codon in exon 8. The intronic sequence may encode a distinct 25-aa fragment of the N-terminal extracellular domain, which could serve as a proposed signal peptide. The continuation of the deduced protein sequence coded by exons 4-13 in SV(1) is identical to that of pituitary GHRH-R. SV(2) may encode a GHRH-R isoform truncated after the second transmembrane domain. Thus SVs of GHRH-Rs have now been identified in human extrapituitary cells. The findings support the view that distinct receptors are expressed on human cancer cells, which may mediate the antiproliferative effect of GHRH antagonists.

L7 ANSWER 10 OF 28 MEDLINE

L7 ANSWER 7 OF 28 MEDLINE
ACCESSION NUMBER: 2001309264 MEDLINE
DOCUMENT NUMBER: 21223623 PubMed ID: 11323691
TITLE: CD45: new jobs for an old acquaintance.
AUTHOR: Penninger J M; Irie-Sasaki J; Sasaki T; Oliveira-dos-Santos A J
CORPORATE SOURCE: Amgen Research Institute and Ontario Cancer Institute, Princess Margaret Hospital, University Health Network, Department of Medical Biophysics, University of Toronto, 620 University Avenue, Toronto, ON M5G 2C1, Canada.. Jpenning@amgen.com
SOURCE: Nat Immunol, (2001 May) 2 (5) 389-96. Ref: 102
Journal code: 100941354. ISSN: 1529-2908.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010604
Last Updated on STN: 20010604
Entered Medline: 20010531

AB Identified as the first and prototypic transmembrane protein tyrosine phosphatase (PTPase), CD45 has been extensively studied for over two decades and is thought to be important for positively regulating antigen-receptor signaling via the dephosphorylation of Src kinases. However, new evidence indicates that CD45 can function as a Janus kinase PTPase that negatively controls cytokine-receptor signaling. A point mutation in CD45, which appears to affect CD45 dimerization, and a genetic polymorphism that affects alternative CD45 splicing are implicated in autoimmunity in mice and multiple sclerosis in humans. CD45 is expressed in multiple isoforms and the modulation of specific CD45 **splice variants** with antibodies can prevent transplant rejections. In addition, loss of CD45 can affect microglia activation in a mouse model for Alzheimer's disease. Thus, CD45 is moving rapidly back into the spotlight as a drug target and central regulator involved in differentiation of multiple hematopoietic cell lineages, autoimmunity and antiviral immunity.

L7 ANSWER 2 OF 28 MEDLINE
ACCESSION NUMBER: 2002124715 MEDLINE
DOCUMENT NUMBER: 21828333 PubMed ID: 11839564
TITLE: Proteolytic cleavage of the CD44 adhesion molecule in multiple human tumors.
AUTHOR: Okamoto Isamu; Tsuiki Hiromasa; Kenyon Lawrence C; Godwin Andrew K; Emlet David R; Holgado-Madruga Marina; Lanham Irene S; Joynes Christopher J; Vo Kim T; Guha Abhijit; Matsumoto Mitsuhiro; Ushio Yukitaka; Saya Hideyuki; Wong Albert J
CORPORATE SOURCE: Department of Microbiology and Immunology, Kimmel Cancer Institute, BLSB 2002, Thomas Jefferson University, 233 S. 10th St., Philadelphia, PA 19107, USA.
CONTRACT NUMBER: CA 51093 (NCI)
CA 69595 (NCI)
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2002 Feb) 160 (2) 441-7. Journal code: 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020226
Last Updated on STN: 20020320
Entered Medline: 20020319

AB Cell surface adhesion molecules are crucial for the development and/or pathogenesis of various diseases including cancer. CD44 has received much interest as a major adhesion molecule that is involved in tumor progression. We have previously demonstrated that the ectodomain of CD44 undergoes proteolytic cleavage by membrane-associated metalloproteases in various tumor cell lines. The remaining membrane-bound CD44 cleavage product can be detected using antibodies against the cytoplasmic domain of CD44 (anti-CD44cyto antibody). However, the cleavage of CD44 in primary human tumors has not been investigated. Using Western blots with anti-CD44cyto antibody to assay human tumor tissues, we show that the CD44 cleavage product can be detected in 58% (42 of 72) of gliomas but not in normal brain. Enhanced CD44 cleavage was also found in 67% (28 of 42) of breast carcinomas, 45% (5 of 11) of non-small cell lung carcinomas, 90% (9 of 10) of colon carcinomas, and 25% (3 of 12) of ovarian carcinomas. Tumors expressing a CD44 **splice variant** showed a significantly higher incidence of enhanced CD44 cleavage. The wide prevalence of CD44 cleavage suggests that it plays an important role in the pathogenesis of human tumors.

L7 ANSWER 3 OF 28 MEDLINE

L7 ANSWER 1 OF 28 MEDLINE
ACCESSION NUMBER: 2002147833 MEDLINE
DOCUMENT NUMBER: 21828651 PubMed ID: 11839669
TITLE: p63 expression profiles in human normal and tumor tissues.
AUTHOR: Di Como Charles J; Urist Marshall J; Babayan Irina; Drobnjak Marija; Hedvat Cyrus V; Teruya-Feldstein Julie; Pohar Kamal; Hoos Axel; Cordon-Cardo Carlos
CORPORATE SOURCE: Division of Molecular Pathology, Department of Pathology, Sloan-Kettering Institute, Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA.
CONTRACT NUMBER: CA 47179 (NCI)
CA 87497 (NCI)
DK 47650 (NIDDK)
SOURCE: CLINICAL CANCER RESEARCH, (2002 Feb) 8 (2) 494-501.
Journal code: 9502500. ISSN: 1078-0432.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020308
Last Updated on STN: 20020529
Entered Medline: 20020528

AB PURPOSE: The p63 gene, located on chromosome 3q27-28, is a member of the p53 gene family. The product encoded by the p63 gene has been reported to be essential for normal development. EXPERIMENTAL DESIGN: In this study, we examined the expression pattern of p63 in human normal and tumor tissues by immunohistochemistry using a monoclonal antibody (clone 4A4) that recognizes all p63 **splice variants**, and by reverse transcription-PCR using isoform-specific primers. RESULTS: We found that p63 expression was restricted to the nucleus, with a nucleoplasmic pattern. We also observed that the expression was restricted to epithelial cells of stratified epithelia, such as skin, esophagus, exocervix, tonsil, and bladder, and to certain subpopulations of basal cells in glandular structures of prostate and breast, as well as in bronchi. Consistent with the phenotype observed in normal tissues, we found that p63 is expressed predominantly in basal cell and squamous cell carcinomas, as well as transitional cell carcinomas, but not in adenocarcinomas, including those of breast and prostate. Interestingly, thymomas expressed high levels of p63. Moreover, a subset of non-Hodgkin's lymphoma was also found to express p63. Using isoform-specific reverse transcription-PCR, we found that thymomas express all isoforms of p63, whereas the non-Hodgkin's lymphoma tended to express the transactivation-competent isoforms. We did not detect p63 expression in a variety of endocrine tumors, germ cell neoplasms, or melanomas. Additionally, soft tissue sarcomas were also found to have undetectable p63 levels. CONCLUSIONS: Our data support a role for p63 in squamous and transitional cell carcinomas, as well as certain lymphomas and thymomas.

L7 ANSWER 2 OF 28 MEDLINE

L16 ANSWER 89 OF 102 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:255524 BIOSIS
DOCUMENT NUMBER: PREV199698811653
TITLE: An 1.5 kb alternative **splice variant** of the platelet derived growth factor alpha-receptor (PDGF alpha-R) as molecular marker for testicular **cancers** of different histogenesis, including carcinoma in situ (CIS.
AUTHOR(S): Oosterhuis, J. W. (1); Gillis, A. J. M. (1); Van Zoelen, E.
E. J.; Looijenga, L. H. J. (1)
CORPORATE SOURCE: (1) Lab. Experimental Patho-Oncology, Dr. Daniel den Hoed Cancer Cent., Academic Hosp., Rotterdam Netherlands
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (1996) Vol. 37, No. 0, pp. 208.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research Washington, D.C., USA April
20-24, 1996
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L16 ANSWER 86 OF 102 MEDLINE DUPLICATE 41
ACCESSION NUMBER: 96226088 MEDLINE
DOCUMENT NUMBER: 96226088 PubMed ID: 8637710
TITLE: Wilms' **tumor** 1 **splice variants** have opposite effects on the tumorigenicity of adenovirus-transformed baby-rat kidney cells.
AUTHOR: Menke A L; Riteco N; van Ham R C; de Bruyne C; Rauscher F J 3rd; van der Eb A J; Jochemsen A G
CORPORATE SOURCE: Lab. of Molecular Carcinogenesis Sylvius Laboratories Leiden University, The Netherlands.
SOURCE: ONCOGENE, (1996 Feb 1) 12 (3) 537-46. Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199607
ENTRY DATE: Entered STN: 19960719 Last Updated on STN: 20000303 Entered Medline: 19960705

L16 ANSWER 74 OF 102 MEDLINE DUPLICATE 33

ACCESSION NUMBER: 97349056 MEDLINE
DOCUMENT NUMBER: 97349056 PubMed ID: 9205060
TITLE: Comparisons of CYP2D messenger RNA **splice variant** profiles in human lung **tumors** and normal tissues.
AUTHOR: Huang Z; Fasco M J; Spivack S; Kaminsky L S
CORPORATE SOURCE: Department of Environmental Health and Toxicology, School of Public Health, University at Albany, State University of New York, 12201, USA.
SOURCE: CANCER RESEARCH, (1997 Jul 1) 57 (13) 2589-92.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970812
Last Updated on STN: 19970812
Entered Medline: 19970725

L16 ANSWER 69 OF 102 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:109221 BIOSIS
DOCUMENT NUMBER: PREV199900109221
TITLE: **Tumor**-susceptibility-gene 101: Characterisation of alternative **splicing variations** in lung **cancer**.
AUTHOR(S): Werner, T. G. (1); Fleischhacker, M. (1); Beinert, T. (1); Jandrig, B.; Sezer, O. (1); Petersen, I.; Witt, C.; Walter, M.; Mergenthaler, H.-G.; Poessinger, K. (1)
CORPORATE SOURCE: (1) Charite Campus Mitte, Med. Klin. mit Schwerpunkt Haematol. und Onkol., Berlin Germany
SOURCE: Annals of Hematology, (1998) Vol. 77, No. SUPPL. 2, pp. S223.
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TITLE: Differential expression of estrogen receptor-beta (ER beta)
in human pituitary **tumors**: functional interactions with ER alpha and a **tumor**-specific **splice variant**.
AUTHOR: Chaidarun S S; Swearingen B; Alexander J M
CORPORATE SOURCE: Department of Medicine, Massachusetts General Hospital and Harvard Medical School, Boston 02114, USA.
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